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DIVINYL ETHER

A REPORT ON ITS USE IN 3,000 CASES

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During the last three years we have used divinyl ether (Vinesthene, May & Baker) on over 3,000 cases and have found it a very useful agent in a variety of circumstances. We have been impressed with its behaviour and its safety and have used it frequently in the teaching of students and interns. We agree with Dawkins [1] who has stated that it is the 'safest known anaesthetic drug'.

We feel that a paper on our experiences with divinyl ether, which is relatively little used in this country, would be of value to the general practitioner and the occasional anaesthetist.

History. Divinyl ether was first prepared by Semmler in 1887, but it was not until 1930 that its anaesthetic properties were discovered by Leake and Chen.[4] In 1933 Gelfan and Bell first described the effects on human beings after administering the drug to themselves.

After their early tests certain disadvantages were noticed. Being extremely volatile, it is likely to cause moisture to freeze in the gas machine or on the anaesthetic mask. A further disadvantage is rapid diminution of its efficiency when exposed to light or air for any length of time. These disadvantages of the pure product are now avoided by the addition of 3.5% absolute alcohol to render it less volatile and 0.01% of a non-volatile oxidation inhibitor, phenyl-anapthylamine; in this form it is marketed under the name of Vinesthene.

Properties. A clear, colourless liquid of specific gravity 0.77 with an odour similar to Ethylene. Children frequently remark that it smells of petrol. It has a boiling point of 28° C (di-ethyl ether being 35° C). It has a slight purplish fluorescence. It is non-irritating to the respiratory tract; molecular weight 70 and vapour density 35. It is highly inflammable and explosive when mixed with certain proportions of air, oxygen and nitrous oxide and should never be used in the presence of diathermy, open flame or unearthed electrical apparatus. We do not use it during bronchoscopy. Divinyl ether is very unstable and when exposed to light or air decomposes rapidly. Heat, too, will cause active decomposition of this drug. Acid accelerates this process with polymerization with the appearance of formaldehyde and formic acid. For these reasons it should be stored in dark-coloured tightly sealed bottles. The proprietary preparation is more stable because of the addition of alcohol and phenyl-anaphthylamine, and may be re-stoppered after use.

Pharmacological Action. The production of the anaesthetic effect is rapid (1–2 minutes). The drug is rapidly eliminated from the body, causing a speedy recovery of consciousness. Muscular relaxation is adequate and relaxation of the masseter muscles allows for easy intubation. There is hardly any respiratory irritation and patients tolerate strong concentrations without objection. Absence of struggling has been a feature of our series, during the induction stage. In the initial stage divinyl ether stimulates the respiratory centre and breathing is deep and regular. If the anaesthetic is pushed, the respiratory centre becomes depressed much more rapidly than does the cardio-vascular centre. Cardiac failure occurs experimentally only as a result of the respiratory depression; in other words, there is always a warning signal if over-dosage is taking place, respiration disappearing before cardio-vascular failure. It should be remembered that the anaesthetic potency of Vinesthene is 4 times that of ether and slightly greater than that of chloroform; therefore only small quantities are necessary for the production of anaesthesia or for marked abdominal relaxation during established anaesthesia with other methods. Goldman [5] has stated that with one ampoule (3 c.c.) of Vinesthene, patients of all ages should be ready for dental work within 55 seconds.

Eye-ball movements may occur even with good relaxation and this fact has been well established in our series. Post-operative nausea and vomiting seldom occur and in a series of 2,600 cases of Vinesthene administration, vomiting more than once during the period of recovery and immediately thereafter occurred in 54 patients. When all patients vomiting a single time, which included a single eructation of mucus, were assessed, the total number was 173 which is slightly more than 6%. This series comprised all out-patient cases where rigid control of diet before operation was not observed. In our own series the immediate occurrence of vomiting following operation was extremely rare; in our non-European series, even less

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common than in European. Post-operative pulmonary sequelae are uncommon according to Hadfield [8] and Henderson.[9] The figure given is about 0.005%. We agree with this finding but our series of cases limited to Vinesthene *alone* is not sufficient to give a satisfactory assessment.

Many authors have criticized this drug's property of producing marked secretion of saliva during induction; we feel that with adequate premedication this finding has not been a disadvantage in our cases and we are convinced that it occurs less frequently than previously reported.

Vinesthene causes no change in the coagulation time of blood and produces no inhibition of peristalsis or uterine contraction. The blood sugar rises at the commencement of anaesthesia but falls much more rapidly than is the case with di-ethyl ether or chloroform. Experimentally in animals no gross pathological changes have been observed in the heart, lungs or kidneys.

Effect of Vinesthene on the Liver. It should be emphasized that this drug should *not* be used as the sole anaesthetic agent for procedures lasting more than 30 minutes. In the presence of anoxia and in long procedures where large quantities of this drug have been used, some evidence of liver damage has been apparent. Oxygen given with Vinesthene minimises this possibility. Experimentally, however, Bourne and Raginsky,[3] using an injection of bromsulphalein (a dye excreted in the liver), found that there was no retention of the dye during anaesthesia. Bourne [12] also applied this experiment to parturient women and found no interference with liver function with Vinesthene whereas with chloroform obvious liver damage was noted. Four fatal cases of acute yellow atrophy have been reported by Dawkins [1] in a series of 35,800 cases. All these cases had anaesthetics for a period lasting from 1–3 hours. In the first instance 180 c.c. was used and in the subsequent cases 275, 355 and 350 c.c. respectively. On the other hand, numerous long anaesthetics have been given with apparent safety.

Convulsions. Convulsions similar to ether convulsions have been reported at about the same frequency as the other ethers. Apparently the same series of factors is usually present as with the ethers, i.e. large doses of atropine, heat in the operating room, pyrexia, young patients, sepsis, carbon-dioxide inbalance, tight harnesses and so forth. So far in our cases we have been fortunate and we have no case in which we can attribute the convulsions to the Vinesthene *per se*. On the other hand, we have had a case of a young African male aged 12 years with a temperature of 104°F, who had septic peritonitis. He was given atropine gr. 1/100 and operated on at 2 p.m. on a hot summer afternoon. He was induced with Vinesthene, nitrous oxide and oxygen and maintained on ether with nitrous oxide and 30% oxygen. He developed a spasm of the glottis before the beginning of the operation; this was followed by twitching in the vicinity of the right eye. This progressed to the left side, followed by the whole face and eventually the limbs. He was taken off ether and given oxygen through an endotracheal tube and 4 c.c. Pentothal Sodium 5% were given intravenously. He settled down within a short period, the operation was successfully performed and he made an uneventful recovery. We feel that there were rather too many factors present in this case (including the use of ether) to attribute the incident to the Vinesthene.

Another type of convulsion has been reported in children during the recovery stages of short anaesthetics:— twitching, cyanosis and unconsciousness has been the picture but recovery has occurred in all these cases.

COMPARISON OF VINESTHENE WITH OTHER ANAESTHETIC DRUGS

Table I compares the most important properties of Vinesthene with those of the other anaesthetic drugs. It is a pleasant drug which may be administered without resistance on the part of the patient as will occur in an ether induction. Induction is very rapid and may be accomplished unlike nitrous oxide in a very high concentration of oxygen. It is non-irritating and may be used to produce all levels of relaxation. It is not safe to use it for very long periods, unlike ether, but for short operations it has the advantage over ethyl chloride in being more practical. It is more powerful than chloroform but has not got the dangerous property of causing cardiac damage.

The advantages of Vinesthene over other anaesthetics therefore are:—

1. Quick induction with good relaxation;
2. Quick recovery;
3. Simplicity of administration;
4. Portable and may be given in the home;
5. Rarely post-operative chest complications;
6. Rarely post-operative nauses and vomiting;
7. The ease with which anaesthesia may be changed to ether.

METHODS OF ADMINISTRATION

It is beyond the scope of this paper to give a detailed description of all forms of apparatus which may be used in the administration of this drug. We feel, however, that it will be of value to mention some of the methods which we have used and our experiences with each.

1. *The Open Method.* Vinesthene is extremely volatile and on opening a bottle and warming it in the hand with a finger over the opening, a stream of Vinesthene may be sprayed on to an open mask in the same way as with ethyl chloride.

A Vinesthene spray attachment, however, may be fitted to the bottle. We rarely use the open mask but find that if necessary a very rapid, pleasant and smooth induction may be obtained in children and adults. A rapid change-over to ether is accomplished without struggling or any difficulty.

2. *Goldman Inhaler and Oxford Inhaler.* These 2 pieces of apparatus are designed for single-dose administrations for short procedures. An ampoule of 3 c.c. Vinesthene is placed in the apparatus (which consists of a bag and face piece) and as the patient breathes in and out anaesthesia is produced which will reach the third stage within about 1 minute. Anaesthesia lasts approximately 1 minute, which is sufficient for very short procedures only.

3. *Goldman Drip Feed.* This piece of apparatus consists of an attachment for the Boyle's apparatus which allows Vinesthene to drop into the mixture of gases at a fixed rate which may be adjusted by turning a knob. This is a delicate piece of apparatus which works very well indeed—but unfortunately it tends to become damaged when used frequently in a busy operating theatre. We used this apparatus continuously for the first 1,000 cases in our series and only gave up its use as we were unable to replace it when it was damaged.

4. *Boyle's Apparatus.* We have now used the chloroform bottle of the Boyle's apparatus for Vinesthene for close on 2 years and found that it works very well indeed. The one point to bear in mind, however, is that only a small concentration of Vinesthene is required and it is very seldom necessary to 'bubble' the oxygen and nitrous oxide through the Vinesthene. The smallest possible concentration given in the usual concentrations of nitrous oxide and oxygen will give fairly good relaxation in the average patient.

TABLE I: COMPARISON OF VINESTHENE WITH OTHER ANAESTHETIC AGENTS;

Property	*Vinesthene*	*Ether*	*Ethyl Chloride*	*Trichlor-ethylene*	*Chloroform*	*Nitrous Oxide*	*Cyclopropane*
Odour	Pleasant	Pungent	Unpleasant	Unpleasant	Sweet, unpleasant	Nil	Very slight
Inflammable	+	+	+	−	−	−	+
Induction	Rapid	Slow	Rapid	Slow	Rapid	Slow without Anoxia	Rapid
Recovery	Rapid	Slow	Rapid	Slow	Very slow	Very rapid	Rapid
Potency	Four times Ether	Potent	Potent	Poor	Less than Vinesthene	Not potent	Very potent
Relaxation	Good	Good	Fair	Poor	Very good	Very poor	Good
Irritation	Nil	Slight	Slight	Slight only	Slight	Nil	Nil
Safety	Very	Very	Fair	Fair	Not safe	Very safe	Fairly safe
Post-operative Nausea ..	Rare	Common	Not common	Common	Common	Nil	Occasional
Post-operative Vomiting ..	Rare	Common	Not common	Common	Common	Nil	Occasional
Salivation	Present—slight	Slight only	Nil	Nil	Nil	Nil	Nil
*Toxicity	Not toxic	Slight	Slight	Slight	Very toxic	Not toxic	Not toxic
*Liver Damage	After half hour	Slight	Undetermined	?No decided view	Common	Nil without Anoxia	Nil
Blood Sugar	Slight rise only	Raised	Raised	Raised	Raised	Not raised	Not raised much
Post-operative complications ..	Rare	Not rare	Not common	Not common	Common	Rare	Occasional
Convulsions	Occasional	Occasional	Nil	Nil	Nil	Nil	Nil
Cardiac Arrhythmias	Nil	Nil	Occasional	Occasional	Occasional	Nil	Common

*Anoxia increases the toxicity and liver damage in all drugs.

5. *Closed Circuit Absorption Apparatus.* We have used Vinesthene in the closed circuit and find it a very valuable adjunct to the anaesthetist's stock-in-trade. We have used it in the ether bottle of the Coxeter-Mushin apparatus and have produced very deep anaesthesia with a minimum of the drug; in fact, we would like to stress the extraordinary depth of relaxation with this method. We have also used it in the chloroform bottle of the Boyle's apparatus when using the Waters canister and find it equally efficient. In our experience with this method, which we have used in 100 abdominal cases, anaesthesia has been deep and relaxation very good indeed without muscle relaxants. Recovery has been rapid and post-operative complications absent. A very small quantity of the drug has been the rule in these cases.

6. *Vinesthene with Air in Obstetrics.* For this purpose the Marret type of inhaler has been used. Vinesthene is placed in a bottle and analgesia may be obtained by the patient's inhaling air which is drawn over the Vinesthene vapour. If the patient inhales too high a concentration and anaesthesia is obtained the administration is stopped by the patient who must cover an air-hole with her finger, as in the Minnit apparatus. If anaesthesia is required, it is a simple matter for the anaesthetist to continue the administration with this method.

7. *Vinesthene Mixed with Ether.* We do not use this method frequently but it may be of value to add Vinesthene to ether to increase its potency in the open mask technique. The usual concentration is 25% Vinesthene with 75% ether. A mixture such as this volatalizes less rapidly than does pure Vinesthene. A mixture of this nature is available under the title 'Vinesthene Anaesthetic Mixture', or 'V.A.M.'. It contains 0.83% absolute ethyl alcohol as a stabilizer.

DISCUSSION

We have administered Vinesthene alone and in combination with other drugs in over 3,000 cases. The youngest patient in the series was a few hours old and the oldest 94 years. We have not limited our series to any particular group but have used it for all types of surgery. We used it as a routine in children and not infrequently in the elderly. We feel that it has a definite place in the 'poor risk' case and in the presence of shock. It is of great assistance for rapid endotracheal intubation and we have found it very satisfactory for blind nasal intubation.

1. *Caesarean Section.* We have anaesthetised over 100 cases for caesarean section using Vinesthene and have had very gratifying results. Our method has been to use nitrous oxide, oxygen, Vinesthene for induction with minimal

Vinesthene, nitrous oxide, oxygen for maintenance, until the uterine wall is incised. At this stage Vinesthene is stopped until the cord is clamped, when Vinesthene and ether are introduced into the circuit. The majority of babies cry immediately on delivery and there is no interference with uterine contraction. We have often had to anaesthetize women for emergency section who have recently partaken of a meal. Rapid induction with Vinesthene enables easy intubation and vomiting is not common. Recovery is rapid and the danger of aspiration of stomach contents is minimized, with the quick return of cough reflex at the completion of the operation.

2. *Obstetrical Procedures.* Internal versions, forceps, and other obstetric manoeuvres have been carried out with good results. Here again deep anaesthesia is obtained without the fear that there will be marked depression of respiration in the baby.

3. *Eclampsia.* For 3 years now we have been trying to convince our obstetrical colleagues of the dangers of the use of chloroform for the control of fits in this condition. Unfortunately we have not been successful. We feel strongly that a drug such as chloroform with its toxic action on the heart, liver and kidneys could well be abandoned for a relatively safe drug without these dangers, as in this condition the kidney and the liver are already damaged. We have not had many eclamptics to deal with, but the few cases that have been controlled with Vinesthene appear to have reacted satisfactorily.

4. *Paediatrics.* We have been using Vinesthene inductions for children for some time now and are convinced of its efficiency, pleasantness and rapid relaxation. We find that the intermittent use of Vinesthene with a nitrous oxide 70% oxygen 30% mixture with minimal ether of very great value in this type of work. In a recent series of 100 consecutive cases at the Transvaal Memorial Hospital for Children we have had no evidence of any post-operative complications using this method. Cases have included minor procedures such as myringotomy to major procedures such as tracheo-oesophageal fistula reconstruction. Age groups in this series were from a few hours to 12 years of age.

Vinesthene with the above method has been of great value in abdominal surgery with the usual induction and nitrous oxide, oxygen, minimal ether maintenance; we have used Vinesthene intermittently:

(a) Just before incision of the peritoneum;
(b) Just before closure.

In these cases the relaxation has been very adequate and recovery of consciousness frequently before the patient leaves the operating theatre.

In plastic surgery of the lip and palate we have used the following technique in over 50 cases under the age of 3 years. Vinesthene induction, intubation with a flexo-metallic tube, maintenance—oxygen, minimal ether and intermittent Vinesthene. The advantage of the latter is that the proportion of ether can be very small as Vinesthene will retain a smooth anaesthetic if the patient lightens, without struggling. Recovery of consciousness occurs at extubation which minimizes the danger of aspiration of blood from the operation site.

5. *Elderly Patients.* In the elderly patient we find Vinesthene of great assistance in induction if Pentothal Sodium is contra-indicated. We are reluctant to use nitrous oxide with anoxia so the addition of Vinesthene to the anaesthetic enables induction to take place with a high concentration of oxygen, thus avoiding anoxia.

6. *Emergency Cases.* In emergency cases where shock, blood loss and full stomachs may be factors of importance, we feel that Vinesthene as an inducing agent may be superior to all other types of anaesthetics. It does not cause the same degree of fall in blood pressure as does Pentothal, and in the presence of blood loss a high oxygen concentration may be of the utmost importance. Rapid intubation, as has been mentioned before, can be accomplished easily and the rarity of vomiting is often of much importance.

7. *Out-Patient Cases (Dental).* For Out-Patient cases, cases of short duration and dental extractions, Vinesthene stands supreme. Recovery is so rapid after short administration with a minimum of after effects, that we feel confident that its use in the Out-Patient Departments of Hospitals should be universal.

8. *Thoracic and Cardiac Surgery.* We have used Vinesthene as a means of induction in 35 thoracotomies in children for various procedures. We have found it of great value in 'wet' lung cases for rapid induction where other drugs are less efficient owing to the inhibition of the drug by much fluid in the respiratory tract. We prefer it here as well, as there is less likelihood of bronchospasm than with Pentothal. In bronchography after bronchoscopy and after the installation of Lipiodol through the bronchoscope in order to produce quiet respiration in children, we have used Vinesthene down the bronchoscope (after the electric light attachment has been removed). This enables us to dispense with Pentothal Sodium and relaxants in these cases.

In thorocoplasty in adults where a local technique is used combined with very light general anaesthesia, the addition of very small quantities of Vinesthene to a nitrous oxide-oxygen mixture will control the cough reflex if necessary. In these cases we find it of advantage to have the patient coughing throughout the procedure, but if control of this factor is needed it may be accomplished with rapidity and efficiency, with the knowledge that the Vinesthene will only act for a short period.

In cardiac surgery where we have had to deal with a patient with cardiac failure with an element of anoxia present, where it has been necessary for a rapid smooth induction without any element of anoxia and a rapid intubation, we have used Vinesthene in preference to other drugs. We dislike Pentothal in these cases because of the fall in blood pressure and a nitrous oxide-ether induction in an adult male is seldom a quiet, steady manoeuvre unattended by struggling and an element of anoxia.

Our procedure in these cases has been a few minutes of pure oxygen followed by the introduction of about 70% nitrous oxide into the circuit. Vinesthene is added slowly and when the patient has passed the second stage ether is introduced *very slowly*.

With this method we have achieved a quiet induction free of struggling with the knowledge that the oxygen concentration has never been depleted. For intubation in these cases we have either pushed our ether or added a muscle relaxant. At this stage, provided the oxygen concentration is high, we do not feel it very important what drugs are used to maintain anaesthesia, provided the

ones directly toxic to the myocardium are avoided. This, too, has been our method in cases of cardiac failure being subjected to other types of surgery.

In oesophageal dilations, where children have to have repeated anaesthetics, we find that using Vinesthene there is seldom the fear that is present when repeated ethyl chloride or intravenous methods are used. The majority of children are asleep before they realize what is happening.

DISADVANTAGES

1. The only real disadvantage of Vinesthene is that it should not be used for long procedures as the sole agent.
2. Vinesthene is very inflammable and explosive and like the other ethers may not be used in the presence of the cautery.
3. It should not be used in the presence of marked liver damage.
4. Vinesthene is very volatile and when used on the open mask is not very economical.

COMMENT

We feel that this substance is of great anaesthetic moment and that it has an important place in modern anaesthesia using physiological methods.

We feel that it could well replace chloroform which is used so very extensively in the smaller centres in this country.

SUMMARY

1. The history of divinyl ether is reviewed.
2. The properties and pharmacological actions are discussed.
3. A comparison is given with other anaesthetic drugs and its advantages are enumerated.
4. Methods of administration are discussed.
5. Various operative procedures are discussed with this drug as the agent used.
6. The disadvantages are listed.

We would like to thank Dr. D. C. Devitt for his help, advice and encouragement in the preparation of this paper.

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ABSTRACT

J. H. Edgcomb, J. Arnold, E. H. Yount Jr., A. S. Alving, L. Eichelberger, G. M. Jeffery, D. E. Eyles and M. D. Young. *Primaquine, SN 13272, a New Curative Agent in* Vivax *Malaria*: A Preliminary Report. J. Nat. Malaria Soc. (1950): **9**, 285.

The authors describe their results in the treatment of non-immune patients suffering from severe infection (induced by 10 mosquito bites) with *P. vivax*, with the combination of Quinine and Primaquine. The latter drug is closely related to Pamaquin, Pentaquine and Isopentaquine; it differs from Pamaquin by having a primary amine substituted for the tertiary terminal amine on the aliphatic side chain in the 8-position of the quinoline nucleus.

This is a very important paper:

1. The authors used the Chesson strain of *P. vivax* which is notorious for its relapsing properties. Thirty-eight patients all relapsed, no matter whether they were treated with Quinine, Quinacrine (Mepacrine), Chlorguanide (Proguanil) or Chloroquine.

2. Ten patients were treated with various doses of Primaquine; out of these only five relapsed, but after increasing the dose to 45 mg. of the base only one out of five patients relapsed.

3. When combining doses of Primaquine base with Quinine, results were as follows. A daily dose of 15 mg. Primaquine caused four patients out of five to relapse, but with a daily dose of 30 mg. none out of five relapsed, toxic symptoms being nearly absent in both instances.

4. Then Quinine was combined with 22.5 mg. Primaquine base daily none out of 10 patients relapsed, no toxic symptoms.

5. Thirteen patients who had been treated earlier with some other drug and relapsed subsequently, were then treated with 22.5 mg. of Primaquine base with Quinine; none relapsed, two suffered from mild, transient, abdominal cramps.

6. Eight patients of the same category as described sub-5, but who were suffering from a second relapse were treated in the same way; one relapsed once again.

7. 240 mg. of Primaquine base probably represents the maximum dose which can be administered with safety for periods longer than a week. The maximum tolerated dose of Pamaquin is 90 mg., of Pentaquine 120 mg., and of Isopentaquine 240 mg.

8. Toxic symptoms are identical to those of Pamaquin and related substances (abdominal cramps, methaemoglobinaemia), but it is of interest to note the effect of Quinine on the production of methaemoglobin. After high doses of Primaquine combined with Quinine methaemoglobin is roughly 50% as great as that formed by the same dose of Primaquine given alone.

9. On an equal weight basis, Primaquine is about four times as active as the best of the other members of the family.

10. As regards the chemotherapeutic index: if that of Pamaquin is 1, that of Primaquine is not less than 10.

11. Another very important fact and one which is beneficiary to the patients is the following. The authors state that 'subsequent studies have shown that 1.64 gm. of the base is in excess of the amount of Quinine needed. A dose of 0.82 gm. of base (1.0 gm. Quinine Sulfate) is certainly sufficient and possibly even as little as 0.547 gm. of base may suffice (⅔ gm. Quinine Sulfate). The smallest effective dose of Quinine has yet to be determined'.

Conclusion: The experiments have shown that *vivax* relapses may be prevented by treating the first attack for two weeks with 1 g. of Quinine Sulfate combined with 22.5 mg. of Primaquine base (= 40 mg. Primaquine diphosphate) daily.

Further experiments will be most welcome to all physicians in malarial regions who have to treat patients with relapsing *vivax* malaria.

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VAN DIE REDAKSIE

BLINDHEID ONDER DIE BANTOE

Dit is dikwels daarop gewys dat die meeste blindheid verhoed kan word. Boshoff [1] het in 1945 gesê dat 95% van die blindheid voorkombaar is, en dat geld wat aan pensioene vir blindes uitbetaal word baie beter bestee kan word in pogings om die siekte te voorkom. In 1948 en in 1949 is daar bv. elke jaar oor die halfmiljoen pond gespandeer op opvoeding, indiensneming en pensioene vir die blindes van alle rasse in die Unie.

Hoewel die blindheidsyfer van die Suid-Afrikaanse Blanke van die laagste in die wêreld is, ding dié van die Bantoe mee vir toelating tot die internasionale kategorie van die allerhoogste blindheidsyfer.[2] In Maart 1950 was daar 22,616 blinde Naturelle aangemeld. Sorsby [3] gee die syfer in Suid-Afrika as 351 Naturelle per 100,000 (teenoor een van 211 vir Kleurlinge en 91 vir Blankes). Sorsby se syfers is, bowendien, waarskynlik konserwatief.

Die oorsake van blindheid onder die Suid-Afrikaanse Bantoe kan aan katarak (43%) en horingvlies-letsels (25%) toegeskryf word. Die oorsaak van horingvlies-letsels is nie bo twyfel nie. Mediese sendelinge het na 'n epidemie van masels horingvlies-letsels opgemerk. Blumenthal,[4] in 'n opsomming van die resultaat van 10 jaar se kliniese studie, spreek die mening uit dat verkeerde voeding numeries die enigste belangrike oorsaak van voorkombare blindheid in die Unie is, veral soos dit die jeug affekteer, en dat die verkeerde diagnose as trachoom van een van die vorms van kerato-conjunctivitis te wyte aan kroniese ondervoeding, algemeen is.

Die mening neem toe, egter, dat die hoë verhouding van horingvlies-letsels aan oogontsteking (d.w.s. bakteriese blindvliesontsteking) en trachoom te wyte is. Die bestaan van trachoom onder Bantoes, hoewel tot nou toe in sommige kringe betwyfel, is onlangs deur Scott *et al.*[5] bewys. Hulle het die insluitsels wat kenmerkend van die siekte is gedemonstreer.

In die afwesigheid van oogontsteking, was trachoom onder die Wes-Afrikaanse Negers as 'n ligte siekte bevind.[6] In Egipte kom epidemies van oogontsteking met tussenpouses voor, gepaard met epidemies van vlieë, wat 'n vinnige verspreiding van oogontsteking veroorsaak wat in sommige gevalle tot seervorming in die horingvlies met komplikasies ontwikkel. Die mening was uitgespreek dat 80% van die blindheid in Egipte die gevolg is van oogontsteking. Die oogontsteking versprei trachoom wat homself

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2. 10de Tweejaarlikse Verslag van die Suid-Afrikaanse Nasionale Raad vir Blindesorg (1948–49): Excelsior Printers (Pretoria) (Pty.) Ltd.
3. Sorsby, A. (1950): Brit. J. Ophthalmol., Supp. XIV.
4. Blumenthal, C. J. (1950): S.-A. Tydskr. Geneesk., **24,** 191.
5. Scott, J. Graham *et al.* (1952): S.-A. Tydskr. Geneesk., **26,** 362.
6. Scott, J. Graham (1945): Brit. J. Ophthalmol., **29,** 244.

EDITORIAL

BLINDNESS IN THE BANTU

It has often been pointed out that most blindness can be prevented. Boshoff [1] stated in 1945 that 95% of blindness was preventable, and that money paid out in pensions for the blind could be much better employed in attempts to prevent the disease. In 1948 and in 1949, e.g. over half a million pounds was spent each year in education, employment and pensions for the blind of all races in the Union.

Although the blind rate in the South African European is amongst the lowest in the world, that of the Bantu competes for admission to the international category of very highest incidence.[2] In March 1950, 22,616 blind Natives were recorded. Sorsby [3] gives the rate in South Africa as 351 Natives per 100,000 (as compared with a rate of 211 for Coloureds and 91 for Europeans). Sorsby's figures, moreover, are probably on the conservative side.

The causes of blindness among the South African Bantu may predominantly be attributed to cataract (43%) and corneal lesions (25%). The cause of corneal lesions is not beyond dispute. Medical missionaries have noted corneal lesions following an epidemic of measles. Blumenthal,[4] summarizing the result of 10 years of clinical study, expressed the view that malnutrition was numerically the only important cause of preventable blindness in the Union, particularly as it affected the young, and that the mis-diagnosis of 'trachoma' for one of the forms of chronic malnutritional keratoconjunctivitis, was common.

A growing body of opinion, however, takes the view that a high proportion of corneal lesion is due to ophthalmia (i.e. bacterial conjunctivitis) and trachoma. The existence of trachoma in the Bantu, though hitherto doubted in some quarters, has recently been proved by Graham Scott *et al.*[5] who demonstrated the inclusion bodies diagnostic of the disease.

In the absence of ophthalmia, trachoma in West African Negroes was found to be a mild disease.[6] In Egypt epidemics of ophthalmia occur seasonally, associated with epidemics of flies which cause a rapid spread of opthalmia, leading in some cases to corneal ulceration with complications. It has been held that 80% of the blindness in Egypt results from ophthalmia. The ophthalmia

1. Boshoff, P. H. (1945): S. Afr. Med. J., **19,** 148.
2. 10th Biennial Report of the South African National Council for the Blind (1948-49): Excelsior Printers (Pretoria) (Pty.) Ltd.
3. Sorsby, A. (1950): Brit. J. Ophthalmol., Supp. XIV.
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5. Scott, J. Graham *et al.* (1952): S. Afr. Med. J., **26,** 362.
6. Scott, J. Graham (1945): Brit. J. Ophthalmol., **29,** 244.

uiteindelik deur die vorming van littekenweefsel in die lid genees. Die littekenweefsel buig die lid en stel sodoende die ooghare in staat om teen die horingvlies te vryf. Uit hierdie reeks gebeurtenisse spruit blindheid dikwels voort.

In soverre as wat trachoom as 'n etiologiese faktor van horingvlies-ondeursigtighede beskou moet word, is dit klaarblyklik belangrik om oogontsteking te behandel en te verhoed. Hiervoor is die grootste enkele maatreël om die bedreiging van vlieë uit te wis en om bronne van besmetting, vir die vlieë om te versprei, uit te skakel.

Baie kan met die gebruik van sulfonamiede gedoen word om oogontsteking te verminder, veral as die middel met die begin van elke vlieë-seisoen gebruik word.[7] Boonop sal vlieë-beheermaatreëls tesame met opvoedingsprogramme oor persoonlike sindelikheid baie bydra om oogontsteking en derhalwe trachoom in die Unie te verminder. So 'n skema moet natuurlik nie geskei wees van die noodsaaklikheid om die gesondheid en voeding van die bevolking wat in gevaar is, in stand te hou nie. Die koste is van minder belang as rekening gehou word met die fantastiese verlies van werkkrag en die ontsettende persoonlike en huishoudelike tragedies waarvoor nie slegs blindheid nie maar selfs belemmerde gesig verantwoordelik is.

7. Lyons, F. Maxwell en Amies, C. R. (1949): Bull. Ophthal. Soc. Egypt

spreads trachoma which eventually heals itself by forming scar tissue in the lid. The scar tissue buckles the lid, thus allowing the eyelashes to rub against the cornea. From this sequence of events, blindness frequently results.

In so far as trachoma must be considered an etiological factor of corneal opacities, it is obviously important to treat and prevent ophthalmia. For this the greatest single measure is to eradicate the fly menace and to eliminate reservoirs of infection for the flies to spread.

Much can be done with the use of sulphonamides to reduce ophthalmia, particularly if the drug is used at the beginning of each fly season.[7] In addition, fly control measures together with educational programmes of personal cleanliness would do much to reduce ophthalmia and therefore trachoma, in the Union. Such a scheme should not, of course, be divorced from the need to maintain the health and nutrition of the population at risk. The price is of small account when considered alongside the fantastic loss of labour power and the dreadful personal and domestic tragedies for which not only blindness but even impaired vision are responsible.

7. Lyons, Maxwell F. and Amies, C. R. (1949): Bull. Ophthal. Soc. Egypt.

A REPORT ON SUCCINYLCHOLINE CHLORIDE (SCOLINE)

AN ULTRA-SHORT-ACTING MUSCLE RELAXANT

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Succinylcholine chloride is a synthetic curarizing agent which differs from the other muscle relaxants in the extreme brevity of its action. The preparation used in this series was supplied by Allen and Hanbury under the name Scoline. It has been found to be a valuable addition to the anaesthetist's armamentarium, enabling him in particular to perform a rapid and easy intubation in all types of cases.

The action of succinylcholine was first reported on by Bovet in France[1] and its pharmacology has been investigated by Castello and de Beer.[2] A short report on its clinical use has been given by Scurr.[3]

Pharmacology. Scoline produces muscle paralysis by depolarizing the motor end-plate. It acts as does acetylcholine, but unlike acetylcholine it cannot be rapidly hydrolysed by cholinesterase. The action potential at the motor end-plate cannot be regenerated and the muscle fibre remains paralysed. Its action is similar to that of decamethonium iodide.

Structurally Scoline resembles 2 molecules of acetylcholine joined together at the α methyl groups. The close similarity of the 2 substances will be seen on comparing their chemical formulae:

$CH_3COOCH_2CH_2N(CH_3)_3OH$
Acetylcholine
$CH_3COOCH_2CH_2N(CH_3)_3Cl$
|
$CH_3COOCH_2CH_2N(CH_3)_3Cl$
Succinylcholine chloride
(Diacetylcholine)

It will also be noted that the quartenary nitrogen atoms in Scoline are separated by a chain of 10 atoms (8 carbon and 2 oxygen), and in this respect it is closely similar to C_{10} (decamethonium iodide).

If Scoline is injected intravenously into an anaesthetized patient, about 20 seconds after the injection, diffuse muscular twitchings occur throughout the body. They are similar to those seen with decamethonium iodide but are more pronounced. They are said to be due to the depolarization occuring at the motor end-plate. They last about 10 seconds and their final disappearance coincides with the onset of muscle paralysis.

Duration of Action. The exact duration of effect was determined in a series of spirometer tracings on anaesthetized patients. A healthy adult patient who had been stabilized in a light plane of anaesthesia with nitrous oxide, oxygen and ether, was given 75 mg. of Scoline intravenously. Twenty seconds after the injection complete apnoea occurred and lasted almost exactly 3 minutes. The apnoea was followed after 3 minutes by the prompt return of deep, rapid respirations. It was found, however, that where the Scoline is given immediately following an induction dose of Thiopentone, the apnoea is somewhat prolonged, lasting perhaps 5 or 6 minutes. A similar effect was noted after Cyclopropane. A second dose of Scoline given 10 minutes after the first

appeared to have no cumulative effect. If, however, Scoline is given shortly after a moderate dose of Flaxedil, the effect of the Scoline is considerably prolonged, and complete apnoea lasting about 10 minutes occurs.

Love [4] reported on 3 cases of prolonged apnoea following the recommended dose of Scoline, one lasting an hour and 2 lasting 20 minutes. Another case has been reported by Harper [5] in which, after 80 mg. of Scoline and 0.7 gm. of Thiopentone, complete apnoea lasting 3 hours occurred. The longest period of apnoea that we have seen has been 10 minutes. In another case, although respirations returned promptly after 5 minutes, intercostal movement was still very much depressed after three-quarters of an hour. A similar prolonged duration of effect rather than degree of effect has been reported with decamethonium iodide.

It is clear from these reports that Scoline can only be given where adequate means for prolonged controlled respiration are at hand.

Dosage. Scoline is supplied in 2 c.c. ampoules each containing 100 mg. Exact dosage is not necessary as its effect is so transient. The recommended doses are as follows:

Large adult: 2 c.c.
Average adult: 1.5 c.c.
Adolescent: 1 c.c.
Children: 0.5–0.75 c.c.

The dose in c.c. may also be calculated as 1% of the body weight in pounds.

Clinical Use. The duration of the effect of Scoline is clearly too short for the surgeon's requirements; its place in anaesthesia lies in its use to establish rapid and easy intubation. It may, however, be used to facilitate sewing up the peritoneum. It is the relaxant of choice in electro-convulsive therapy and may also be used successfully for difficult orthopaedic manipulations. Over the last 3 months we have used it extensively for intubations in tonsillectomies, dental cases and also for oesophagoscopy and bronchoscopy.

Technique. As Scoline is rapidly hydrolysed by alkaline Thiopentone, it must be injected intravenously from a different syringe, although the same needle may be used. We have found a 3-way tap very satisfactory for this purpose. Twice the sleep dose of Thiopentone is given, and this is immediately followed by an appropriate dose of Scoline. The lungs are inflated for a few moments with oxygen and an endotracheal tube lubricated with liquid paraffin is passed either blindly or under direct vision. The relaxation is most profound, the cords are usually wide open and little or no reaction to the tube occurs. The lungs are then inflated with nitrous oxide, oxygen and ether. It is important that the ether should be introduced rapidly by manual compression of the bag as the effect of the Scoline quickly passes off and the patient will begin to react to the tube. Trilene has been used but has been found less satisfactory than ether. Thiopentone and Scoline alone may be used but it is easier to keep the laryngeal reflexes quiescent by the addition of ether. Where the operation is away from the mouth, the larynx may first be sprayed with 2% Decicaine or the tube lubricated with 10% Nupercaine ointment.

This technique has been found to be very satisfactory for oesophagoscopy and has also been used for bronchoscopy in dry cases. The ether is omitted and additional doses of Thiopentone and Scoline are given to keep the patient quiescent. The lungs are inflated with oxygen through the side tube of the bronchoscope.

For electro-convulsive therapy smaller doses are given. In the average adult 30–50 mg. is adequate. The patients are premedicated with Atropine gr. 1/50, a small dose of Thiopentone is given, and this is immediately followed by the Scoline. We have used Scoline without Thiopentone in a number of cases. The very unpleasant sensations of Scoline in the conscious patient appear to be covered by the retrograde amnesia caused by the shock therapy. The Scoline is injected and the shock given immediately the muscular twitchings cease. The lungs are then inflated with oxygen until normal respirations begin.

No untoward reactions have been seen. There is no appreciable effect on the cardiovascular system and E.C.G. tracings are normal. The somewhat disturbing tachycardia so often seen with Flaxedil from vagal inhibition does not occur. Scoline, like decamethonium iodide, probably has little or no effect upon either the sympathetic or vagal ganglia. No increased bleeding from the tonsillar bed has been observed. Bleeding times taken before and after the injection of Scoline have been normal.

Effects of Prostigmine. Prostigmine is a very effective antidote to Flaxedil but its use with Scoline is contra-indicated as it produces an enhanced effect. If a patient is given 1 mg. of Prostigmine shortly before the Scoline is injected, complete apnoea for 10 minutes occurs.

Castello and de Beer have shown in animal experiments that the prolonged action produced by Prostigmine is due at least in part to the inhibition by Prostigmine of certain substances normally responsible for the rapid destruction of Scoline.

SUMMARY

The use of Scoline in a series of about 100 cases is described. It has been found to be a valuable drug giving a short period of profound relaxation lasting as a rule between 3 to 5 minutes, and during which intubation may be performed with ease. Its use in such short cases as tonsillectomy, dental work, bronchoscopy and oesophagoscopy is described. The method used was to give twice the sleep dose of Thiopentone followed immediately by the Scoline through a 3-way tap. After inflation of the lungs with oxygen the patient is intubated and ether is introduced rapidly by manual compression of the bag (except in the case of bronchoscopy, where further doses of Thiopentone and Scoline are given). It has been found to be the relaxant of choice in electro-convulsive therapy. Smaller doses of Thiopentone and Scoline are given. Means by which the patient may be inflated with oxygen must always be at hand.

Prostigmine produces an enhanced effect but no antidote is necessary as the effect of Scoline is so transient. Cases have, however, been reported in which the effect has been prolonged. These cases are treated by inflation of the lungs with oxygen until normal respirations begin. No other untoward reactions have been found to occur.

We wish to thank Dr. S. Disler, Medical Superintendent, Grey's Hospital, for permission to publish these cases and also Dr. M. Ginsburg, Acting Medical Superintendent, Town Hill Mental Hospital, Pietermaritzburg, for his invaluable assistance in the electro-convulsive therapy.

We also wish to express our sincere thanks to the surgical staff, Grey's Hospital, for their unfailing co-operation.

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TWO CASES OF VENOUS STASIS

RADIOLOGICAL FINDINGS

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Two cases of venous stasis are described, one being secondary to mediastinal obstruction and the other due to an anomaly of the venous system.

This report demonstrates the use of contrast media in the diagnosis of vascular pathology. The nature of the pathological changes is discussed as fully as possible.

CASE 1

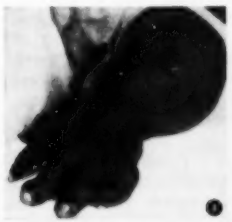
A Native male aged 30 years was admitted to King Edward VIII Hospital in January 1950 complaining of swelling since birth of the left forearm and hand.

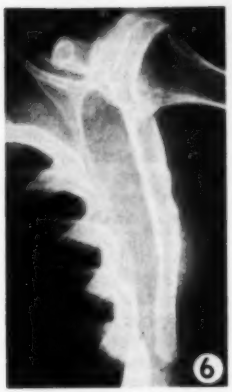
He stated that the arm had been painful for the last year but, following a blow on the arm 2 weeks before admission, the pain had become worse. The left hand had always sweated profusely. Biopsies had been performed and the patient told that he had an abnormality of the blood vessels.

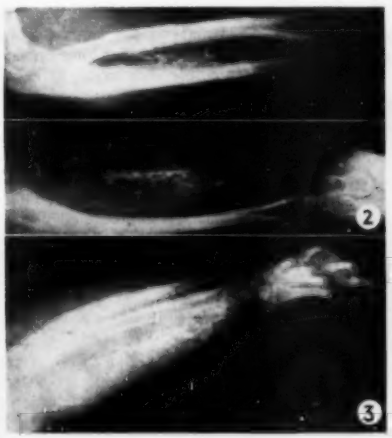
Examination. The left forearm and hand were grossly enlarged (Fig 1), with localized bluish, non-pulsatile swellings, having a soft rubbery consistency. The fingers were perspiring profusely and the limb was slightly warmer than its fellow. Three further swellings were present, one located in the left pectoral region, one below the left axillary fold and one on the lower part of the left chest wall posteriorly.

The cardiovascular system, the respiratory system and the central nervous systems were normal.

Radiological Report: January 1950. 'There is considerable swelling of the soft tissues of the forearm and the hand (Figs. 2 and 3). Lying anteriorly in the soft tissues is an irregular mass of calcified tissue scattered over all areas, but especially







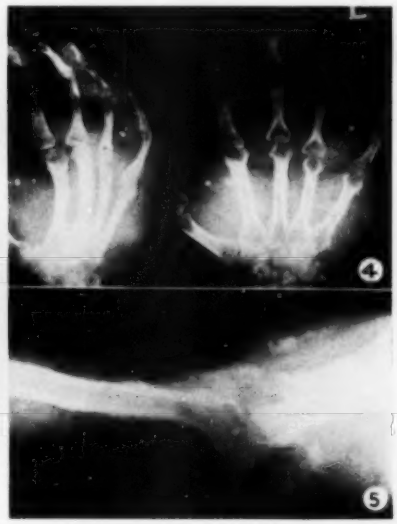


Fig. 1. Hand and arm showing gross swelling.
Fig. 2. Radiograph of left forearm (first admission 1950). Early tree-like vascular calcification in the soft tissues of the flexor aspect of the forearm. Sclerotic changes in radius and ulna.
Fig. 3. Radiograph of left forearm (second admission 1951). Considerable increase of soft tissue swelling. Vascular calcification and proliferative bony change.
Fig. 4. Radiograph of the left hand. Soft tissue swelling and scattered phleboliths. Sclerotic changes in the metacarpals.
Fig. 5. Radiograph of the left elbow. Bony changes in the radius and ulna and the lower third of the humerus. Vascular calcification in the soft tissues of the forearm. Phleboliths in the soft tissues of the upper arm.
Fig. 6. Radiograph of left shoulder. Pressure erosion on the lateral border of the scapula.

in the hand, where numerous small densities are seen. The shafts of both the radius and ulna are irregular in contour and density and, especially at the distal ends, there is a lacy periosteal reaction. No definite joint pathology is seen. There is relative decalcification about the bone ends in the hand and the trabeculation is coarse in these regions.

'The appearances are those of a vascular tumour, probably of a haemangiomatous nature, but the bone changes are marked and the unusual periosteal reaction raises a suspicion of malignancy.'

The patient refused amputation and was discharged. He was re-admitted in July 1951 suffering from considerably more pain and showing great increase in the size of the arm.

The following investigations were done.

Blood Count:
Hb.: 12.95 gm. %.
White cells: 7,000 per c.mm.
Polymorphs, 47%; lymphocytes, 40%; monocytes, 4%; eosinophils, 9%.

Radiological Examination: 'The soft tissue swelling of the left forearm and hand (Figs. 4 and 5) has increased considerably since the previous radiographs in 1950. The soft tissue calcification has also increased in extent. The lower third of the left upper arm is also visibly swollen. The calcification has a tree-like vascular pattern. Small oval areas of calcification are seen in the region of the hand. There is a suggestion of erosion of the lateral margin of the scapula immediately beneath the glenoid (Fig. 6). Stippled calcification of the soft tissues beneath the scapula is demonstrated.'

Conclusion. 'The appearance is consistent with the presence of an extensive angiomatous malformation of the vessels of the left arm and chest wall. Numerous areas of calcification are present and appear to lie within the walls of the vessels and possibly within the lumen as well. The findings confirm the previous radiographic impression and demonstrate the progressive nature of the lesion.'

Venography. Diododrast was injected into one of the dilated veins on the dorsum of the hand (Figs. 7 and 8).

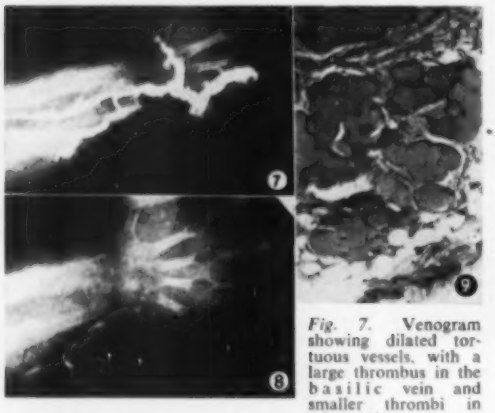


Fig. 7. Venogram showing dilated tortuous vessels, with a large thrombus in the basilic vein and smaller thrombi in other veins.

Fig. 8. Venogram 6 minutes after injection of dye, showing residual dye in an almost completely occluded basilic vein.
Fig. 9. Photomicrograph showing thin-walled, dilated venous channels.

This outlined an abnormally large and tortuous metacarpal vein, proceeding proximally through the dorsal venous network to where normally the median ante-brachial vein is situated. Here 2 main channels were apparent, one of which probably communicated with the deep venae comites. Medially an enormously dilated, deformed and calcified vein was present in the position of the basilic vein. This showed stasis with pooling of blood, 6 minutes after injection. The cephalic vein was not outlined.

Biopsies: *Lymph Node* (*Sinus Catarrh*). The lymph node shows prominent sinuses infiltrated by numerous large mononuclear cells and recent haemorrhage. There is a marked siderosis—possibly part of a generalized siderosis. No tumour tissue is present and the features are non-specific.

Left Arm. Fibro-fatty tissue infiltrated by cavernous haemangiomatous tissue (Fig. 9). There are deposits of haemosiderin and focal collections of chronic inflammatory cells. A small vein shows calcification in the wall. Another vessel shows fibrous occlusion. There is no evidence of malignancy.

Left Chest Wall. The fragment from the chest wall consists of fibro-fatty tissue with numerous dilated thin-walled vascular channels and several small arteries. There is no evidence of malignancy.

DISCUSSION

When this case was first seen, a vascular anomaly was immediately suspected. The proliferative bone changes raised the possibility of a malignant lesion. On re-admission it was noted that the bone changes had become more extensive, as had the areas of vascular calcification. More phleboliths were present and investigation of the left chest wall showed soft tissue swelling, erosion of the lateral margin of the scapula and scattered phleboliths. Bony changes now involved the distal end of the left humerus. Venography confirmed the suspicion of dilated abnormal venous channels and marked stasis. Many of the vessels showed long linear defects in the dye columns, compatible with areas of extensive thrombosis. This was particularly well marked in the case of the basilic vein, dye still enveloping a large thrombus some 6 minutes after injection.

Similar changes were presumably present in the upper arm and the left chest wall, as worm-like masses of vessels were easily palpable. The conclusion was that the appearances represented a diffuse congenital phlebectasia.

Thompson and Shafer [1] mention that the condition was first described by Bockenheimer [2] in detail. They state that the diffuse systemic haemangioma described by Allen, Barker and Hines [3] closely resembles phlebectasia. They also quote the detailed studies of Sabin [4] in which it is emphasized that blood vessels spread over the developing embryo in definite sheets of capillaries, and that abnormal development of veins and arteries from this plexus appears to afford the chief explanation of vascular anomalies. The opinion of McNealy [5] and of De Takats,[6] that the type of vascular anomaly depends on the stage in which embryologic arrest occured, is also quoted.

In the clinical description Thompson and Shafer [1] mention that skin changes in the nature of atrophy and ulceration may occur. An increase in skin temperature, though less than that found in arteriovenous aneurysm, is sometimes present. They state that changes in osseous growth such as hypertrophy or atrophy may occur. They do not, however, indicate whether the hypertrophy and atrophy mentioned represent increased or decreased growth of normal bone, or whether there are unusual changes in bone density or new bone formation. Their

statement that muscular tissue becomes atrophic because of disuse or from pressure from the adjacent vascular mass, is comprehensible, and is well demonstrated in our case where little or no muscle tissue can be present. The biopsy reports indicate that no muscle tissue is present and that fibro-fatty changes were observed. It is, of course, possible that the section was not taken from a muscle-containing area.

In Case 1 the increased sclerosis of bone and the proliferative periosteal reaction has already been commented on. Whilst this was originally thought to indicate a malignant lesion, it soon became apparent that chronic venous congestion was probably the etiological factor. Baker [7] quotes Pommer's theory (1885) that chronic venous congestion results in increased tissue pressure in the medullary spaces of bone. This is presumably produced by vascular overfilling, or by an increase of the cellular or fluid content of the soft tissues confined within the bone spaces. The increased tissue pressure in turn stimulates osteoclastic activity resulting in osteoporosis. Although Baker considers that this theory is probably the most comprehensive, he states that there have been many case reports where venous stasis has resulted in marked osteosclerosis and not osteoporosis.

We feel that our case is yet another example of this particular type of change. Thompson and Shafer [1] do not mention calcification of vessels, which is a striking feature of the case described above. The absence of comments on both this feature and the sclerotic proliferative bony changes is probably due to the fact that the cases seen by these authors were in an early stage. The plates demonstrated in their paper certainly suggest that the vascular changes are far less marked than in our case. It is also possible that trauma in our patient might well have resulted in massive haemorrhage. This could quite easily have precipitated or accelerated the changes seen on the radiographs. It is significant that, from the time of trauma, the patient experienced increased pain, and noted a progressive and more rapid increase in size of the limb than in the preceding years. Well-marked hyperhidrosis is present and was found in Thompson's cases.

As the condition appears to be closely related to the cavernous type of haemangioma, X-ray therapy may be of considerable value. The forearm is, however, probably too extensively involved for any satisfactory response to radiation. Irradiation of the upper arm and the chest wall might obtain results. If a cure is not obtained, it should tend to decrease the risk of extensive haemorrhage during amputation of the arm at the level of the mid-humerus. Amputation would appear to be the only method of treatment of the grossly enlarged and useless forearm.

CASE 2

A Native male aged 45 years was admitted to the King Edward Hospital with a diagnosis of upper mediastinal syndrome. He complained of dyspnoea, non-productive cough and swelling of the face and neck.

Examination showed engorgement and slight oedema of the face and neck. The neck veins were distended and not pulsating, distension being greater on the right than on the left side.

The heart was not clinically enlarged and no abnormal pulsation was seen or felt. The sounds were normal though faint and no murmur was heard. The blood pressure was 140/96 mm. Hg in the right arm and 136/94 mm. Hg in the left arm. No tracheal tug was felt.

The chest, abdomen and nervous systems were normal. A moderate amount of albumin was present in the urine.

Investigations:

Blood Count. Hb. 12.2 gm. %. White cells: 3,000 per c.mm. Polymorphs, 35%; lymphocytes, 63%; monocytes, 2%.

The *Wassermann reaction* was positive.

Four examinations of sputum were negative for tubercle bacilli.

Radiological Report on the Chest. There is a rounded density present in the second right interspace (Fig. 10) with heavy streaking through the hilum. The mediastinum appears slightly widened. The heart appears normal. The appearances are in favour of early re-infective tuberculous infiltration.

Screening of the Chest. The density in the second interspace appears to be situated anteriorly. The mediastinal widening is apparently due to the aorta, and the appearance is suspicious of specific aortitis.

Angiocardiogram. The dye flowed to the commencement of the right innominate vein where a complete block was demonstrated. From this region the dye proceeded through a complicated mass of dilated vessels, which communicated with the 2 superior intercostal veins and the azygos vein, the direction of the blood flow being reversed. From the azygos vein the blood passed to the lower intercostals and lumbar azygos veins, thence to the inferior vena cava and the heart. The heart appeared to be normal though the filling on the left side was poor. The obstruction apparently extended from the origin of the right innominate vein into the superior vena cava as far as its entry into the pericardium. The cause of the obstruction was not demonstrated, but was thought to be thrombotic, secondary to intrinsic disease, possibly syphilis. Secondary invasion by neoplasm or other pathology could not be excluded.

DISCUSSION

This case demonstrated the effect of occlusion of the superior vena cava. Oedema and cyanosis of face, neck and upper extremities are pathognomonic. The venous pressures were not measured, but it was noted that the insertion of a cannula into an arm vein, before injecting Diodrast, resulted in a venous blood return, very dark in colour, spurting out as if under increased pressure.

Anatomy. The middle mediastinum contains the pericardium and heart, the first portion of the aorta, the pulmonary artery, part of the superior vena cava, the phrenic nerves, the hila of the lungs, bronchial lymph nodes and the termination of the vena azygos major. The latter communicates with the lumbar veins and occasionally the left renal vein and the inferior vena cava. At times it may act as a collateral shunt in the presence of obstructive lesions. In the case described it fulfilled this role.

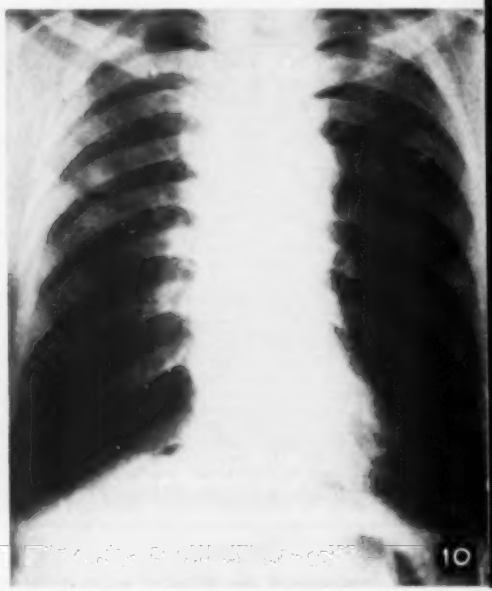
Superior vena caval obstruction, originally thought to be rare, is now known to be not uncommon.

In 1934 Ehrlich, Ballon and Graham [8] collected and analysed 309 cases. Oschner and Dixon [9] reviewed the literature adding 2 cases of their own. Roberts, Dotter and Steinberg [10] studied 55 cases in 1951. McIntyre and Sykes [11] quote 502 cases in 1949. Hussy Katz and Yater [12] reported 35 cases in 1946.

From the literature it would appear that tumours of the thorax are important factors in superior vena caval obstruction.

Malignant thoracic tumours, either primary or metastatic

in origin, are quoted as accounting for from 33%-78% of the cases of superior vena caval obstruction. Other cases mentioned are syphilis, external compression, non-malignant growths and fibrosis following mediastinitis.



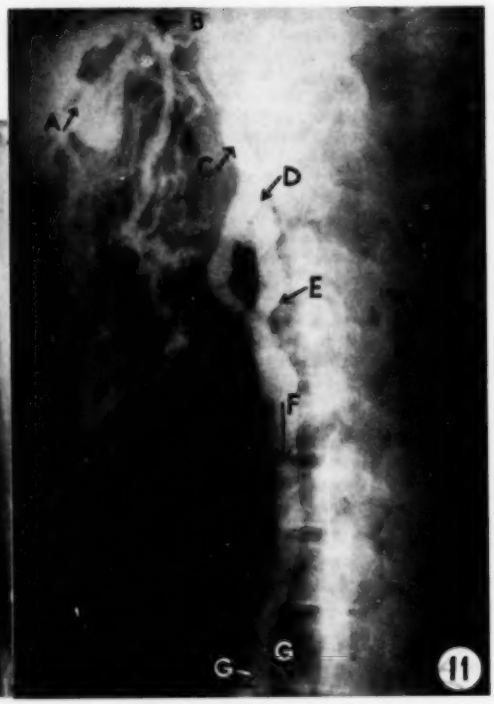


Fig. 10. P-A. view of the chest showing slight widening of the mediastinum. An opacity in the second right interspace. Nature not proven.
Fig. 11. Angiocardiogram. Print reversed. Annotated plate showing filling of the azygos and the hemiazygos veins.

A. Subclavian.
B. Jugulars.
C. Superior intercostal artery.
D. Left superior intercostal.
E. Superior hemiazygos.
F. Azygos vein.
G. Inferior hemiazygos veins.

The cause of the obstruction in the present case was not determined. Syphilis was thought to be likely in view of the positive Wassermann reaction and the appearance of the aorta on screening, but in view of the high incidence of tumours in the published figures, this possibility could not be excluded. Unfortunately no final diagnosis was made, as the patient left hospital prematurely. We understand that he has since died, but no post-mortem findings are available.

We wish to record our thanks to Dr. J. Parker, Superintendent of the King Edward VIII Hospital, Durban, for permission to publish these case reports; to Dr. J. Wainright and Miss M. McClaggen for the histological studies and the photographic reproductions respectively.

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ENDEMIC GOITRE IN SOUTH AFRICA

H. MALHERBE, M.A. (CANTAB.), M.B., B.CH. (RAND)

South African Institute for Medical Research, Johannesburg

Greenwald [1] has taken South Africa to task for her failure to investigate fully endemic goitre as it occurs here. Referring to the original discovery in 1929 of goitre in the Langkloof, and to subsequent investigations which could not establish a relationship between the iodine content of foodstuffs and the incidence of the disease in this part of the country, he states: 'Here was an almost ideal situation for a careful epidemiologic investigation. The opportunity was neglected'. It must be admitted that more could have been done to study the condition here. The following facts briefly outline the work that has been undertaken.

In 1929 the Department of Public Health reported [2] the occurrence of goitre in the Langkloof (Uniondale district) and in the Pretoria district. District Surgeons throughout the Union were asked to report on the incidence of goitre in their areas; and provision was made for the supply of iodized salt on request in affected parts of the country.[3]

In the same year, the Cape Department of Education published [4] the results of a survey it had made of over 8,000 children during the previous few years. Although areas of high endemicity were not revealed, it was concluded that the Province showed a wide distribution of subgoitrous thyroid enlargement.

The next report came in 1931, when the Department of Public Health announced [5] that goitre occurred in a valley in the Rustenburg hills in the Transvaal; and there followed in 1932 Frack's description [6] of the Kosterfontein valley near Rustenburg, where 18 out of 24 families were affected by goitre.

About this time, Blom [7, 8] was carrying out iodine estimations on various human and animal foods at Onderstepoort. Buttner [9] suggested in 1935 that lack of sunlight in the sheltered valleys of the Langkloof might cause a metabolic disturbance leading to failure of the body to utilize iodine; but this was refuted by Schur Brown [10] who pointed out that the amount of sunlight in the Langkloof was not as limited as Buttner had stated. In 1938 Steyn [11] suggested that fluorosis might play a part in the pathogenesis of goitre, and mentioned having seen cases of goitre in the Kenhardt district, North-western Cape, where fluorine was commonly found in the natural water.

Kark in 1944 reported [12] that he had encountered goitre at Witzieshoek on the Orange Free State side of the Drakensberg during a nutrition survey of Bantu children, and mentioned having found it also at Underberg, Polela and Impendhle, in Natal. He stated that Dormer had investigated goitre at Estcourt in 1940, but this study remains unpublished.

In 1945 the National Nutrition Council asked all magistrates and local authorities in the Union to submit information regarding the occurrence of goitre in their districts; but this did not lead to much increase in knowledge of the distribution of the disease, and the matter was left in abeyance.

Malherbe and Osburn, in 1946, observed and studied goitre in the Nelspruit district of the Eastern Transvaal, where it occurred among both Europeans and Bantu. Acting on a suggestion made in a personal communication by Dr. Frack that goitre might be endemic as far west as Zeerust, they included this area in their investigation, and several endemic foci were found there. In order to provide a standard with which goitrous populations might be compared, they examined 7,000 children in Johannesburg; and the results of comparisons with certain groups of country children have been published.[13]

The Report of the Chief Medical Inspector of Schools for Natal in 1948 [14] presents some figures based on the work done in 29 schools by Dr. C. Strydom; but a full survey has not been undertaken in Natal. For the purpose of investigating endemic areas and analysing food and water samples, there has been formed at Pretoria University a Goitre Research Committee under the chairmanship of Prof. D. G. Steyn; and during the past few years several areas, including the Langkloof, the North-western Cape and Swaziland have been visited. In 1950 this Committee made a recommendation to the Minister of Health that all salt should be iodized.

Greenwald has objected to the iodine-lack theory of the causation of simple goitre, and there is evidence to show that iodine intake is not necessarily reduced in endemic parts. Possibly it is the utilization of iodine that is more at fault, demanding a higher intake than normal to produce satisfactory functioning of the gland. But the factor responsible for the primary dysfunction of the thyroid still remains unknown, and presents an interesting problem. The provision of increased amounts of iodine for consumption by the whole population would almost entirely eliminate simple goitre from this country, but it would not give an answer to the question: What causes simple goitre?

There are two reasonable approaches to the problem of endemic goitre. One is to provide iodized salt, thereby suppressing the disease while treatment is maintained. The other is to make exhaustive researches to trace the unknown etiological factor. The former is simple and practical, but intellectually unsatisfying. The latter course may take years to produce an answer, during which time many persons would continue to suffer the discomforts and dangers of goitre.

It is here suggested that the two methods should be combined, so that the problem may be treated as a whole. The provision of 10 parts of potassium iodide per million parts of salt, replacing entirely ordinary salt, would probably suffice to suppress the appearance of goitre in this country; but in order to estimate the success of any form of treatment, the incidence of goitre should first be established by means of a co-ordinated survey, and regular examinations should subsequently be made during the administration of iodine. Once the incidence has been determined, analyses of food, soil and water, together with animal experiments, could proceed in order to detect differences between areas of high and low endemicity. The occurrence of goitre in children provides a reliable and readily available indication of the incidence in a wider population; and if we could co-ordinate our treatment, our research and our regular examination of school-

children, we would be contributing much to the health of the nation.

SUMMARY

A brief outline is presented of investigations into the distribution and causation of simple goitre in South Africa since the first report in 1929 of its occurrence here.

It is suggested that the incidence of goitre should first be determined by a Union-wide survey, and that subsequently the iodization of all salt, the regular examination of school-children, and further researches into the etiology of endemic goitre should proceed simultaneously.

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HYPERTENSIVE ANGINA TREATED WITH CYCLOSPASMOL

REPORT ON A CASE

CARL T. H. BARRETT, M.B., CH.B. (LEEDS)

Johannesburg

Mrs. E.M.S. aged 61 has suffered from typical hypertensive angina since 1945. Her blood pressure was 180/110 mm. Hg, and an X-ray revealed left ventricular enlargement. The attacks were controlled by Tab. Glyceryl Trinitrate gr. 1/200.

In October, 1948, she suffered a coronary thrombosis. An ECG showed an anterior infarct of minimal degree. The final ECG showed complete recovery; screening of the chest, showed no further ventricular enlargement as compared with the X-ray taken in 1945.

The attacks of angina were now no longer associated with exertion and in 1949 she was put on a salt-free diet and injections of 2 c.c. Mercuhydrin bi-weekly. The blood pressure was reduced from 180/110 mm. Hg to 140/100 mm. Hg. The anginal attacks were reduced in number and severity. The improvement lasted for 2 months only. The blood pressure rose to its former level and the attacks of angina became more frequent. In addition to the administration of Aminophyllin and Phenobarbitone, by mouth, large doses of vitamin E, Nicotinic Acid and Priscol were tried without benefit.

Most attacks responded to Tab. Glyceryl Trinitrate gr. 1/100 or amyl nitrite 3-minim capsules. Occasionally Pethidine by injection was required to relieve the pain. At this time, numerous extrasystoles occurred which responded excellently to Belladenal. The patient was then given propyl thiouracil, commencing with 600 mg. daily and gradually reducing to 200 mgm daily. The response was dramatic, but after one month, painful ulcers of the tongue appeared which necessitated withholding the drug. Frequent white cell counts were performed and all were satisfactory. After the ulcers had healed, the propyl thiouracil was again given with much benefit, but again after 3–4 weeks the ulcers appeared and only cleared up when the thiouracil was discontinued. A further course of thiouracil produced the ulcers and this line of treatment was dropped.

In May 1950, the patient had another thrombosis, this time a small posterior infarct, from which she made a good recovery. The blood pressure did not return to the original level of 180/110 mm. Hg. The readings showed wide fluctuations, the highest being 150/95 mm. Hg and the lowest 100/70 mm. Hg. Despite the semi-invalid life led, the patient suffered from frequent attacks of pain, from her 'coronary insufficiency'.

In October 1950, in an effort to relieve this pain, thoracoscopy was performed and phenol injected into the thoracic sympathic on both sides from T2–T5. This did not produce much freedom from attacks of pain, but the blood pressure became less fluctuant.

In the hope of reproducing the benefit given by propyl thiouracil, a total of 86.2 m.c. of radio-active iodine was administered over the period April–September 1951. There was no material improvement.

In November, 1951, experimental supplies of Cyclospasmol became available. Treatment was commenced with 1 pill (20 mg.) *t.d.s.* increasing to 2 *t.d.s.* when improvement was first noticeable. The attacks of angina became less frequent and less severe. The systolic blood pressure was lowered by 30 mm. Hg and the diastolic by 20 mm. Hg. The dosage was reduced slowly until a maintenance dose of one pill daily was reached. After one month on this maintenance dose the attacks became more frequent and the dosage was increased to 2 *t.d.s.* with improvement. The only side-effect noticed was that the patient complained of abdominal distension. A blood count, taken after the patient had been taking the drug for 2 months, showed no alteration from her normal count.

In April 1952, the patient had an emotional upset and the attacks of pain became stronger and more frequent despite taking the Cyclospasmol. The dosage was then increased to 4 *t.d.s.* with great improvement. There was no further depression of blood pressure with this large dosage, and no new side-effects.

ASSOCIATION NEWS : VERENIGINGSNUUS

GRIQUALAND WEST BRANCH: JULY MEETING

A meeting was held on Thursday, 17 July 1952, in the Board Room of the Kimberley Hospital. Dr. S. Perel was in the Chair and 19 members attended.

Two very interesting films on the *Kidney Function in Health and in Disease* were shown (by courtesy of Eli Lilly & Co.).

The meeting terminated with a vote of thanks to the firm's representatives, the projectionist and the Chair.

The monthly meeting was held on Thursday, 31 July 1952. Dr. S. Perel was in the Chair and 17 members attended.

A symposium on *Fractures of the Neck of the Femur* was presented. *Dr. J. Botha* opened the discussion by describing the aetiology and types of fractures encountered.

Mr. A. B. de Villiers Minnaar discussed the pathology of fractures in this region with particular reference to aseptic necrosis of bone.

Dr. J. E. Vaughan Jones gave a short description of methods of treatment, past and present.

Mr. J. Visser discussed the prognosis and the social aspect.

Several very interesting X-ray pictures were then demonstrated by Mr. Minnaar and Mr. Kretzmar, and questions were posed by several members.

The meeting terminated with a vote of thanks to the Chair.

L. Schrire,
Honorary Secretary.

14 August 1952.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

SOUTHERN MEDICAL AID SOCIETY

The Executive Committee of the Federal Council has agreed to the re-instatement of the Southern Medical Aid Society as an approved society, to operate on the *Tariff of Fees for Approved Medical Aid Societies* as from 1 September 1952.

L. M. Marchand,
Assistant Secretary.

Medical House,
Cape Town.
30 August 1952.

SOUTHERN MEDIESE HULPVERENIGING

Die Uitvoerende Komitee van die Federale Raad het die herstel van die Southern Mediese Hulpvereniging as 'n goedgekeurde mediese hulpvereniging, onderhewig aan die *Tarief vir Mediese Hulpverenigings*, vanaf 1 September 1952 goedgekeur.

L. M. Marchand,
Assistent Sekretaris.

Mediese Huis,
Kaapstad.
30 Augustus 1952.

PASSING EVENTS

In the recently published Report of the latest session of the *Expert Committee on Biological Standardization* held in Geneva in December 1951, the Appendix contains a complete and up-to-date list of all the current International Biological Standards. Most of these are of many years' standing, but some are quite new, having been adopted at the 1951 meeting.

* * * *

The Annual Report of the Cape Town Mothers' Clinics has recently been published. All the workers in this organization are voluntary. The least privileged members of the community, irrespective of race or creed, benefit as a result of these Clinics. The services of the medical profession are appreciated, because the profession has been very sympathetic towards the activities of the Clinics ever since these were started nearly 21 years ago.

The routine gynaecological examination of new cases, and annual re-examination of old cases, have assisted the doctors in improving the health of the mothers. Those requiring operative or other treatment have been referred to specialists at hospitals and to other institutions. The Committee feels that this constructive medical aspect of the work should be stressed as, by improving the health of the mother, the condition of the family as a whole is also greatly improved.

Those interested in the work of the Clinics should communicate with the President, Mrs. R. L. Scott, *Bizana*, Plumstead, C.P.

* * * *

On Wednesday, 10 September, *The Problem of Suffering* will be discussed at a general meeting of the Medical Christian Fellowship. The meeting will be held at 8 p.m. in the Physiology Theatre at the Medical School, Mowbray, C.P. All interested practitioners are invited to attend.

REVIEWS OF BOOKS

ARCHITECTURE OF ARTHRODESIS

Architectural Principles in Arthrodesis. By H. A. Brittain, *O.B.E.*, M.A., M.Ch., F.R.C.S. (Pp. 196 + vii, with 257 illustrations. 42s.) Second Edition. Edinburgh: E. & S. Livingstone Ltd.

Contents: 1. Indications for Arthrodesis. 2. Causes of Failure of Arthrodesis. 3. Architectural Principles. 4. Bone Grafts from the Tibia. 5. Ischiofemoral Arthrodesis. 6. V-Arthrodesis of the Hip. 7. Arthrodesis of the Symphysis Pubis. 8. Arthrodesis of the Knee. 9. Arthrodesis of the Ankle. 10. Arthrodesis of the Spine. 11. Arthrodesis of the Interphalangeal Joints of the Thumb and Finger. 12. Arthrodesis of the Carpo-Metacarpal Joint of the Thumb. 13. Arthrodesis of the Wrist. 14. Arthrodesis of the Elbow. 15. Posterior Scapulohumeral Arthrodesis of the Shoulder. 16. Appendix—The Naughton Dunn Stabilization of the Foot. 17. Index.

It is with great pleasure that one sees that a second edition of this very well-known book has now been published, 10 years after the first. In this the author has adopted the interesting device of including pictures taken about 11 years later, of the original cases published. These indicate how excellent are the results of the methods founded upon his architectural principles, and how well they have stood up to the test of time. In particular, the results of ischio-femoral arthrodesis, which has proved such a boon in the surgery of the hip, are most striking. It is interesting to note that the author still considers the original lateral approach for this operation preferable to and easier than the posterior approach now used by many surgeons.

The architectural principles are 4 in number and, wherever possible, the method of arthrodesis is adapted to comply with these, which are:

1. The graft should be placed with its long axis in compression rather than in tension.

2. The breadth of the graft should be placed in the position of maximum stress.

3. Where possible a joint should be locked by 2 grafts crossing each other in the shape of the letter X.

4. There should be adequate protection of the graft.

The author has included a detailed description of the classical operation of Naughton Dunn of triple arthrodesis of the foot, which gives such excellent results in providing a stable, painless weight-bearing foot.

The book is beautifully produced and is a credit both to the author and the publishers.

ELECTROENCEPHALOGRAPHY

Diagnostic Electroencephalography. By Hans Strauss, M.D., Mortimer Ostow, M.D., Med.Sc.D. and Louis Greenstein, M.D. (Pp. 282 + xiii. $7.75.) New York: Grune & Stratton.

Contents: Part I—General Aspects of the Electroencephalogram. 1. Definition. 2. History. 3. Equipment. 4. Preparation for the Recording. 5. The Recording. 6. Artefacts. 7. Components of the Electroencephalogram. 8. The Normal Electroencephalogram. 9. Physiology of the Electroencephalogram. 10. Psychic Function and the Electroencephalogram. 11. Classification of Normal Electroencephalograms. 12. Metabolic Changes and the Electroencephalogram. 13. Development of the Human Electroencephalogram. 14. The Abnormal Electroencephalogram. 15. Survey of Classification of Records and Symbols Used. 16. Provocative Tests. 17. Frequency Analysis.

Part II—The Electroencephalogram in Disease. 1. Intracranial Tumors. 2. Supratentorial Gliogenous and Metastatic Tumors. 3. Supratentorial Meningiomata. 4. Cerebellar Tumors. 5. Tumors of the Cerebellopontine Angle. 6. Other Posterior Fossa Tumors. 7. Pituitary and Suprasellar Tumors. 8. Other Midline Tumors. 9. Other Intracranial Extracerebral Tumors and Tumors of the Cranium. 10. Chronic Sundural Hematoma. 11. Gumma. 12. Chronic Cystic Arachnoiditis. 13. Cerebrovascular Disease. 14. Transient Hemiplegia. 15. Arterial Hypertension. 16. Subarachnoid Hemorrhage. 17. Subarachnoid Hemorrhage Arising from Angiomatous Malformations at the Convexity. 18. Aneurysms at the Base of the Brain without Hemorrhage. 19. Syphilis of the Central Nervous System. 20. Encephalitis. 21. Meningitis. 22. Cases with Acute Sinus and Middle Ear Disease and No Evidence of Intradural Disease. 23. Epidural Abscess. 24. Brain Abscess. 25. Chorea Minor. 26. Cerebral Trauma. 27. Multiple Sclerosis. 28. Senile Dementia. 29. Presenile Dementia. 30. Huntington's Chorea. 31. Wilson's Disease and Pseudosclerosis. 32. Dystonia Musculorum Deformans. 33. Degenerative Diseases of the Central Nervous System Affecting the Cerebrum. 34. Anoxemic Encephalopathy. 35. Congenital or Early Acquired Cerebral Defects. 36. Tuberous Sclerosis. 37. Oxycephaly. 38. Amaurotic Family Idiocy. 39. Amyotrophic Lateral Sclerosis. 40. Meniere's Syndrome. 41. Labyrinthine Vertigo. 42. Migraine and Histamine Headaches. 43. Diseases of the Spinal Cord. 44. Miscellaneous Muscular Syndromes. 45. Diseases of the Cranial and Spinal Nerves and Roots. 46. Epilepsy. 47. Narcolepsy. 48. Attacks of Apnea. 49. Attacks of Syncope. 50. Carotid Sinus Hypersensitivity. 51. Disorders of the Carbohydrate Metabolism. 52. Disorders Arising Outside the Central Nervous System. 53. Exotoxic Disturbances of Cerebral Activity. 54. The Electroencephalogram and Mental Illness.

Part III—The Diagnostic Evaluation of the Electroencephalogram. 1. Description of the Record. 2. Classification of the Record. 3. Physiologic Interpretation. 4. Diagnostic Interpretation. 5. The Localizing Interpretation of the Record. 6. The Report. Bibliography. Illustrations. Index.

Although the literature on electroencephalography has assumed vast proportions and many thousands of papers have been published on the subject, there has, until recent times (with a single exception) been no readily available source book of reference to which anyone interested could turn for information and enlightenment. Within the last year or two a few very satisfactory volumes have been published and the authors of this book are to be congratulated on a worthy addition to a not undistinguished company.

The book is the outcome of a great deal of experience by 3 well-known clinical electroencephalographers and shows evidence of much critical thought and deliberation, though one may find occasion not to agree with all their conclusions. They have, for example, laid down criteria of normality which are extremely rigid (certainly much more so than any other workers) and they refuse to consider as abnormal many a type of record that most electroencephalographers would accept as not normal, even though there might be differing opinions about the degree of abnormality present and its significance. Now it is quite true that there is too often a tendency to 'over-interpret' a record and to read into it an abnormality that will not stand the test of critical judgment, but one is inclined to conclude that the authors have veered too strongly in the other direction, though it may in many ways be a preferable move. As a result of this standard their figures in many instances differ from other published results and, for example, the 'non-specific dysrhythmias' find no place in their classification. Again one can question their summary dismissal of 'flicker stimulation' as an aid only in experimental electrophysiology and of no value in clinical work.

Despite these criticisms it is a most valuable work, enhanced by good reproductions of records, some excellent tables under various headings, and a good bibliography. Every electroencephalographer and neurologist should add this book to his personal library and any medical practitioner who wishes to inform himself about this important field of medical investigation could do no better than to get this book.

It is scarcely necessary to add that the publishers have printed and bound it in excellent fashion.

CORRESPONDENCE

W.C.A. FEES: THEIR NEED FOR REVISION

To the Editor: Further to the letter of Dr. W. Blignaut (28 June 1952, p. 540) I wish to add some quotations and remarks.

From the 'Accident Fund: Assessment Rates 1951-52', *Handbook of the W.C.A. Fund*, p. 3, sect. 2, *Assessments*:

(1) '. . . Should a surplus arise, it would be *returned to employers* either by means of rate reductions or by means of 3-yearly rebates.'

You will note the employer is the one to profit by surpluses, not the medical profession.

The same booklet (p. 7, sect. 7), *Rates*:

(2) 'The rates are designed to produce sufficient revenue to meet the costs of accidents . . . and will be adjusted from time to time to produce the required income for each year *and nothing more*.'

(3) From the *Medical Handbook* of the Act as amended by Act 27 of 1945, 2nd ed., p. 5.: 'Payment for medical aid shall be in accordance with the scale prescribed . . . after consultation with the Medical Association of South Africa. . . .'

(4) From the *South African Medical Journal*, 26 July 1952: *Standards of Social Security and Practising Doctors: A Statement by the World Medical Association*, p. 615: 'IX. Remuneration of medical services ought not to depend directly on the financial condition of the insurance organization.'

(5). 'XII. There shall be no exploitation of the physician, the physician's services or the public by any person or organization.'

Comment: Will some member of the Medical Association of South Africa with whom the W.C.A. Commissioner has consulted, kindly reconcile quotations (4) and (5) with quotation (3)—and a consideration of the Scale of Fees prescribed? I do not doubt that all members of the Medical Association will consider the principles of (4) and (5) as just.

Now to descend to specific cases. This town has a Mission Hospital with its own X-ray apparatus. The nearest Government-owned hospital is 66 miles away. No local practitioner either in this or the surrounding towns has his own private X-ray apparatus. The W.C.A. administrators refuse to pay any doctor's fee for diagnostic X-rays; they will only pay the hospital's fee (the hospital has no resident doctor or radiologist, so the doctor must do his own X-ray diagnosis) 'because no specific provision for payment of medical fees under these circumstances is laid down in the Scale of Fees'. The hospital is entitled to its fees, and so is the doctor entitled to fees for the exercise of his technical skill in X-ray diagnosis.

This cannot be an isolated instance and it is time it was rectified.

Another grumble—the employees of the Provincial Roads Department in this area are very loath to report Workmen's Compensation cases as such and will avoid doing so if they can because of the long delays before they are paid out. This must cause a lot of additional work and delays at Headquarters—and Headquarters is to blame. I have known of cases where doctor's and hospital fees were paid 2 years after the original accident.

I have just received a cheque for 16s. from the Commissioner in payment for 1 consultation and 8 fl. oz. medicine and one visit at 3.30 a.m. with intravenous injection of coramine and intracardiac injection of adrenaline and coramine in an attempt to save a workman's life. Surely I am entitled to more than this?

J. W. D. Paisley.

P.O. Box 31,
Cala, C.P.
30 July 1952.

MARRIAGE AND HEALTH CERTIFICATES

To the Editor: Am I right in assuming that Switzerland and Sweden are the only 2 countries in which the law compels marrying couples to undergo an examination previous to marriage and to furnish a certificate of health, i.e. freedom from venereal disease, tuberculosis and mental disease? Are there any other countries (including South Africa) where such a law exists or is contemplated? If not, why not? I would be glad to hear from any of your readers.

Inquirer.

12 August 1952.

The Medical Association of South Africa
Die Mediese Vereniging van Suid-Afrika

AGENCY DEPARTMENT : AGENTSKAP-AFDELING

JOHANNESBURG

Medical House, 5 Esselen Street. Telephones 44-9134-5, 44-0817
Mediese Huis, Esselenstraat 5. Telefone 44-9134-5, 44-0817

PRAKTYKE TE KOOP : PRACTICES FOR SALE

(Pr/S34) Progressive Transvaal town dispensing practice. Average gross income £3,500 p.a. Excellent surgical facilities. Owner going overseas.

(Pr/S39) Pretoria practice. Gross annual income, £3,200 to £3,500. Premium required £1,750. No house for sale. Full details on application.

(Pr/S43) Bloemfontein. Exceptionally well-established solus prescribing practice. Average annual receipts approx. £7,000. Premium required £4,250. Great deal of midwifery done. Practice offers great scope for practitioner with surgical ability.

(Pr/S46) O.F.S. dispensing practice. R.M.O. and M.O.H. appointments. Average monthly takings £260. House to let at £10 p.m. Premium required £1,000, which includes instruments, drugs and furniture. Cash is preferred, but terms could be discussed.

(Pr/S51) Transvaal hospital town dispensing practice. Gross income over £6,000 per annum. It is essential that this practice be worked by two men, one to be a surgeon. Premium required £3,500, and terms could be arranged. Practice can only be sold if house and surgery are bought for cash. Details on application.

(Pr/S48) Northern Rhodesia. Unopposed solus dispensing practice. Annual gross takings £5,000 (cash £3,500 and accounts £1,500). No bad debts, very little night work. Premium required £1,600. Drugs and furniture on valuation. Surgery buildings for sale or for hire. Will suit doctor who is not interested in city life.

(Pr/S52) Progressive Transvaal hospital town. Practice with excellent scope for expansion. Premium required £600 and terms could be arranged. Premium includes drugs, furniture and instruments valued at £160.

(Pr/S54) Established branch practice in Johannesburg. Annual income £1,000. Premium required £500. Very much scope for expansion.

(Pr/S55) Well-established practice in northern suburbs of Johannesburg. Will suit an English-speaking doctor. Premium required £1,000. Full details on application.

* * * *

DURBAN

112 Medical Centre, Field Street. Telephone 24049

PRACTICES FOR SALE : PRAKTYKE TE KOOP

(PD10) General practice, Natal inland city. European and non-European patients. Scope for midwifery and surgery. Premium required £1,250, cash preferred, but terms will be considered. For immediate sale.

(PD13) Natal Lower South Coast practice, near Pondoland border, suitable for retired doctor. Area developing and large Police holiday camp in vicinity. Excellent climate and very good fishing. Premium required £400, includes good stock of drugs and dressings, instruments and dispensary furniture. House for sale £1,800, including stand of one-third morgen. Bond available. For immediate sale. Owner having taken a full-time appointment.

LOCUM REQUIRED

From 27 August to 21 September. Natal village, 25 miles from Stanger. £2 2s. per day, all found, plus £5 car allowance. Woman doctor preferred, but must possess own car. District Surgeoncy and Native practice. No surgery or midwifery, and no night work.

Natal Midlands village. Month of November. £2 12s. 6d. per day, free board and lodging. Petrol and oil supplied. Single man preferred, but not essential. Mixed country general practice. No midwifery or major surgery. Hardly any night work. Dispensing of stock mixtures only. Native interpreter employed.

KAAPSTAD : CAPE TOWN

Posbus 643, Telefoon 2-6177 : P.O. Box 643, Telephone 2-6177

PRAKTYKE TE KOOP : PRACTICES FOR SALE

(1003) Transkei. Well-established dispensing practice. Total cash receipts 1951, £3,311. D.S. and M.O.H. appointments. Large well-built house for sale at £3,300. Premium required £1,500. Loan for £3,000 available.

(1010) Cape Town. Nucleus of practice with excellent scope for expansion. Average annual receipts £1,100. Premium required, £850, which includes drugs, few instruments, half-share furniture. Consulting rooms shared with specialist.

(1016) Eastern Province. Unopposed solus practice. Average annual receipts £2,471. Premium for goodwill £1,000. Drugs, furniture and instruments offered at £190. Terms available. Attractive modern home to rent at £8 10s. p.m. Rental roomy surgery, £3 p.m.

(992) South-Eastern Cape hospital town. Premium required £1,500, which includes drugs, furniture and instruments worth approximately £1,350. Flat plus surgery to let at £6 p.m. Gross average annual cash takings, £2,500. Easy terms. Owner wishes to specialize.

(1101) Coastal City. Better-class general practice. Gross annual receipts £2,200. Premium required £1,750. Terms possible. Practice is expanding.

(1099) Well-established unopposed East Griqualand practice. Three good appointments. House to let at nominal rental. Gross cash takings for year ending December 1951 were £3,668. Premium required, £2,150. Terms available. Excellent opportunity for English-speaking doctor.

(746) Large dispensing practice, mainly non-European. Average annual cash receipts approx. £5,200. £5,500 required for premium, drugs and surgery furniture. Details on application.

(895) Partnership share in practice of Specialist Physician. Details on application.

(1115) Cape Town suburban practice. Details on application.

City of Cape Town

MEDICAL SUPERINTENDENT OF HOSPITALS

Applications are invited from registered medical practitioners under 45 years of age for the position of Medical Superintendent of Hospitals in the City Health Department.

The successful applicant will be required to devote the whole of his time to the work of the Council and will not be permitted to engage in private practice.

The duties comprise the administration and clinical responsibility of the City Infectious Diseases Hospital, Portswood Road, Cape Town; the Brooklyn Hospital for Chest Diseases (including Formidable Epidemic Diseases) and the Langa Hospital. In addition to the above duties the successful applicant will be required to carry out such other medical duties as may be allocated to him from time to time by the Medical Officer of Health.

Applicants must state their qualifications and submit full details of experience in the modern treatment of Infectious Diseases, Tuberculosis and Venereal Diseases and in addition should state their experience in the administration of large hospitals.

The salary will be at the rate of £1,620 per annum in Grade 152, Scale £1,620 × 60—£1,920, plus a temporary cost-of-living allowance. The successful applicant will be required to reside at the City Infectious Diseases Hospital where accommodation, light, fuel and water will be provided, for which at present a deduction of £165 per annum is made from the above emoluments.

The appointment will be subject to the provisions of the Municipal Ordinance No. 19 of 1951; to the Standing Orders of the Council and to the Municipal Staff Code, all as amended from time to time. Furthermore, the salary grade assigned to the position is subject to the approval of the Minister of Health.

Applications in duplicate on the prescribed forms obtainable from the Senior Staff Officer, Municipal Buildings, Longmarket Street, Cape Town, should reach him not later than noon on 30 September 1952.

M. B. Williams
Town Clerk
10264

City Hall
Cape Town
6 September 1952
6702

South African Railways and Harbours Sick Fund

APPOINTMENT OF RAILWAY MEDICAL OFFICER: PORT ELIZABETH: DISTRICT 'D'

Applications are invited from registered medical practitioners for the position of Railway Medical Officer, Port Elizabeth District 'D', at a salary of £1,173 per annum, plus £50 per annum workshops allowance, plus the fees and allowances prescribed by the Regulations of the Sick Fund and with the right of private practice.

The salary will be subject to adjustment in accordance with the census of members to be taken on 1 April of each year.

The appointment will be made in terms of the Regulations of the Fund, and will be subject to termination on 4 months' notice being given by either side.

The successful candidate will be required to reside at Port Elizabeth, to take up the appointment on a date to be arranged, and to carry out his duties in accordance with the Regulations of the Fund.

Applications should reach the District Secretary, Cape Midland District Sick Fund Board, 116 Mutual Arcade, Port Elizabeth, not later than 20 October 1952, and should state:—

1. Full name.
2. Qualifications (when and where obtained).
3. Experience (when and where obtained).
4. Date of birth.
5. Country of birth.
6. Whether married or single.
7. Whether fully bilingual.
8. Whether South African citizen.
9. What Government appointment, if any, is held.

Canvassing by or on behalf of any applicant is liable to disqualify such applicant.

Any further particulars may be obtained from the District Secretary at the above address, on application.

Johannesburg P. J. Klem
6 September 1952 *General Secretary*

Natal Provincial Administration

VACANCY: SENIOR MEDICAL OFFICER: ADDINGTON HOSPITAL

Applications are invited from registered medical practitioners for appointment to a vacant post in the Casualty Department.

Appointment is on 12 months' contract and the salary attaching to the post is as follows:—

Two years' service after qualification: £400 p.a., plus privileges.

Three years' service after qualification: £600 p.a., plus free quarters or an allowance in lieu thereof.

Four years' service after qualification: £700 p.a., plus free quarters or an allowance in lieu thereof.

Five or more years' service after qualification: £800 p.a., plus free quarters or an allowance in lieu thereof.

In addition to the foregoing salary, a temporary cost-of-living allowance is also payable.

Applications giving full details of experience and qualifications, should reach the Director of Provincial Medical and Health Services, P.O. Box 20, Pietermaritzburg, by 15 September 1952. AD7135

South African Forge Medical Benefit Society

P.O. BOX 321, GERMISTON

Medical practitioners are invited to register as panel doctors with the above Benefit Society, which has been approved by the East Rand Branch of the Medical Association of South Africa.

Conditions of service may be obtained from, and applications should be addressed to, the Secretary at the above address.

Applications will be considered if received within one month of the publication of this notice.

Post Wanted

Doctor seeks post as assistant in Cape Town area from about November. Write 'A. M. O.', P.O. Box 643, Cape Town.

Printed by Cape Times Ltd., Parow, and Published by the Proprietors, THE MEDICAL ASSOCIATION OF SOUTH AFRICA, MEDICAL HOUSE, 35 Wale Street, Cape Town. P.O. Box 643. Telephone 2-6177. Telegrams: 'Medical'